REMARKS

By the subject amendment, Applicant has cancelled Claims 1-7 without prejudice and amended Claim 8 to better define the invention. More specifically, Claim 8 is recast as a method for preventing microtubule assembly in a human carcinoma cell by selective disruption of ß-tubulin function in turn leading to the prevention of microtubule assembly and leading to cell breakdown. Claim 9 is new, dependent on Claim 8 and specifically directed to treating of human breast carcinoma. Support for human breast carcinoma was acknowledged in the Office Action (Paper 13). Claim 10 is new, dependent on Claim 8 and specifically directed to treating a human patient. Support stems from experiments using human carcinoma cell lines.

Full support for Claim 8, as amended, is found in the originally filed disclosure, for example:

- Page 1:

"The present invention is also concerned with a method of selectively attacking cancer cell key proteins by providing derivatives of 1-aryl-3-(2-chloroethyl)ureas having specific spatial configurations allowing these derivatives to dock inside cells at pre-selected sites."

- Page 7:

"The inventors have surprisingly found clear evidence that the CEUs of the present invention are potent antimicrotubule agents that covalently bind to ß-tubulin and consequently prevent microtubule assembly";

- Page 8:

"Western blot analysis confirmed the disruption of microtubules and evidenced the formation of an additional immunoreactive β-tubulin with an apparent lower molecular weight on SDS polyacrylamide gel. Incubation of MDA-MB-231 cells with [urea-^{14}C]-4-tBCEU revealed the presence of a radioactive protein which coincided with the additional β-tubulin band, indicating that CEU could covalently bind to the β-tubulin. The 4-tBCEU-binding site on β-tubulin was identified by competition of the CEU with colchicine, vinblastine and iodoacetamide, a specific alkylating agent of sulfhydryl groups of cysteine residues. Colchicine, but not vinblastine, prevented formation of the additional β-tubulin band, suggesting that 4-tBCEU alkylates either Cys239 or Cys354 on β-tubulin."

- Fig. 2 specifically demonstrates microtubule depolymerization;

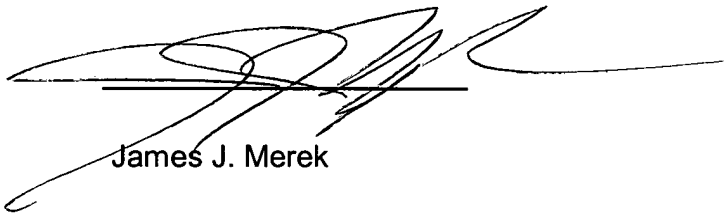
- Fig. 6 and 7 specifically demonstrate the selectivity of the compounds of the present invention towards microtubule assembly disruption.

- Since multiple strains of two types of cancer cell lines (wild-type chinese hamster ovary and human breast carcinoma) were extensively tested with positive results, it is clear that the originally filed specification provides support for the claimed feature of treating various types of human carcinoma.

It is respectfully submitted that Claims 8-10, as amended, are neither taught nor rendered obvious by the prior art cited by the Examiner. The prior art thus far clearly falls short of providing or even predicting a method for selectively causing microtubule assembly disruption in a cancer cell. The present invention is clearly beneficial when treating cancer patients since the compounds used in the method of Claims 8-10 specifically act on cancer cell protein sites and thereby avoid detrimental drug therapy side effects of commonly used chemotherapy agents.

In light of the foregoing, favorable reconsideration of this application is earnestly solicited. It is respectfully requested that the subject patent application be expeditiously passed to issuance without delay. It is believed that no fee is due. However, should that determination be incorrect, the Patent Office Officials are hereby authorized to charge any deficiencies to Deposit Account No. 13-2759. The undersigned requests to be notified of any charges to the aforementioned deposit account. Should the Examiner have any questions or wish to discuss further this matter, please contact the undersigned at telephone number provided below.

Respectfully submitted,



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